

Viruses, HPV and Kissing: A Neglected Social Habit

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Abstract

There are many microbes that can be transmitted by kissing. These include: Herpes Simplex Virus (HSV), Epstein Barr Virus (EBV), Cytomegalo Virus (CMV), Meningococcal disease, Respiratory Infectious viruses and Human Papilloma Virus. Of these the HPV is oncogenic. There are 448 types of HPV, many of which assist in predisposing to-, or precipitating-, neoplastic change. Much research about the various types of HPV, shows which are antigenic and/or oncogenic. Discussed here are fundamental facts about common infectious microbes in saliva, stressing HPV modes of transmission and how kissing, with other intimate social attitudes and behaviors impact the spread of HPV.

Keywords: viruses; hpv; kissing; habit

Background

Kissing, (Oral-to-oral) is common and globally widespread. Although kissing is innocently used as a sign of affection, respect, intimacy or eroticism, kissing also constitutes mechanisms for unobtrusive microbial exchange and disease transmission. There are at least 6 types of microbes that can be transmitted by kissing. These include: Herpes Simplex Virus (HSV), Epstein Barr Virus (EBV), Cytomegalo Virus (CMV), Meningococcal disease, Respiratory Infectious viruses and Human Papilloma Virus. HSV's causes lip genital and gingival morbidity; EBV is known a "Kissing disease" and cause infectious mononucleosis, fatigue, sore throat and swollen lymph glands. CMV often affects pregnant women and immunocompromised individuals, Meningococcal causes meningitis with serious morbidity and mortality outcomes, Respiratory Viruses, like Covid, Bird Flu, Zaka Viruses, is spread by droplets of infectious mucus and saliva. With Oral-to-Genital contact (Oral sex) commonly transmitted diseases include: HPV, HSV-1 and HSV-2, Gonorrhea, Syphilis, Chlamydia, Trichomonas, Hepatitis A, B and C, HIV, and others. Of these the HPV infection predisposes to-, and can cause oncogenic change [1,2]. Cancer has a major influence on society worldwide affecting millions and their healthcare. Many researchers strive to "find a cure for cancer" by understanding and/or deconstructing underlying oncogenic causes and consequent cellular mechanisms.

Introduction

Much attention has been directed to the Human Papilloma Virus (HPV) and its relevance to Cancer.

Prior to the introduction of HPV vaccines, the prevalence infected people with HPV in the USA population by age 45 years, among those with at least one opposite sex partner, was as high as 80-90 percent [2,3]. There are 448 types of HPV, many of which assist in predisposing to-, or precipitating neoplastic change. The classification of the HPV viruses is constructed on different DNA sequences. There are five genera of HPV: (i) alpha, (ii) beta, (iii) gamma, (iv) mu, and (v) nu, which are divided into low-risk HPV (LR-HPV), and high-risk (HR-HPV) subtypes [3,4]. From among the 448 types of HPV, 15 of them are grouped as oncogenic: HPV16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 68, 73, and 82. Also types 26, 53, and 66 are deemed to be potentially carcinogenic. HPV16 and HPV18 display the highest oncogenic potential although regional genetic diversity moderates their pathogenicity [2,3,4]. Comparisons of the HPV16 long control region (LCR) sequences, five groups are separated: European (E), Asian (As), Asian-American (AA), African 1 (Af-1), and African 2 (Af-2) [5,6,7]. The most ubiquitous types of HPV found in other geographical regions were HPV-16, 18, 52, 31, 58, 39, 51, and 56, with the frequency of genotypes varying in different regions. Overall HPV infection is more present in African nations of penury compared to more affluent countries. Eastern Africa and sub-Saharan Africa have a prevalence between 34% and 24.0%. Eastern Europe has a prevalence at 20% and West Asia is as low as 1.7%. [2]. North America has a smaller range of HPV-genotypes, yet there is a high diversity of HPV genotypes in Asia [8,9]. HPV of genitalia infection in males increases after age 15 years, and globally, nearly one in three

men are infected with HPV of some type, with approximately one in five men having one or more high-risk (HR) HPV types [10]. Recent research implicates HPV infection in the occurrence of further types of cancer, other than uro-genital cancers [11]. While HPV is mainly regarded as mainly a sexually transmitted disease, (spread by contact during sexual intercourse), other unexpected modes of transmission are also possible but too often ignored.

Aim

Discussed here are fundamental facts about HPV with focus on various forms of kissing, social behaviors, attitudes and oral sexual practices which facilitate HPV spread.

Current

In the 21st Century among the most important understanding of oncogenesis was the realization that chronic infection with specific HPV genotypes were a cause of cervical neoplasia. Extensive epidemiology, supported by molecular, microbial, genetic and clinical, research yielded positive significant proof that cervical cancer derives from chronic, untreated infection by certain HPV genotypes. Accordingly, it is evident that cervical cancer is the result of HPV viral infection. This cause-and-effect stresses that prophylactic vaccinations are necessary and important as preventative therapy against developing cervical-, and /or genital cancers, and other associated diseases caused by HPV infections [2,3,13]. Yet HPVs are stringently species specific, which property indicates that human papillomaviruses (HPVs) only infect humans. Jumping species is not known or recorded. Epithelia with inherent squamous-cell maturation are tropism-targets for the double stranded DNA HPV viruses, and after completing their maturation cycle in fully differentiated squamous epithelium, HPVs are infective. Different combinations and/or sequential infections by oncogenic types cause neoplastic changes from mild, moderate and severe dysplasia, to frank neoplastic change. Among the most significant breakthroughs in recent cancer research has been the finding of consistent and persistent infection with select HPV genotypes as the cause-and-effect in cervical cancers. Extensive epidemiological, virology, medical, clinical molecular research studies have confirmed the evidence that cervical cancer results from chronic infection by specific HPV genotypes [3,9,10]. Accordingly, because affirmation that cervical cancer is the outcome of HPV viral infections,

it is apparent that the urgency and necessity for recognizing that vaccination is now a vital and significant strategy for prophylaxis. All strategies for prevention have become important. Primary prevention of many cancers and other diseases deriving from HPV, is now (2025) more imperative than ever [3,9,10,16].

Discussion

Since the realization of oncogenic properties were exposed, stress moved away from treating local lesions, like genital warts. To ensure patients were successfully vaccinated against developing other morbidities, they had to be inoculated before their sexual debut [11]. This realization was known to the medical profession, but rarely appreciated by the public in general [13]. Also, some religious disciplines preached celibacy, and for some time young boys were not inoculated. Both of these latter approaches were failures, and vaccinations for all boys and girls regardless of faith, were exhorted to be vaccinated against HPV at-, or just after-, onset of puberty [13]. A popular anti-Covid-vaccine movement constrained the implementation of HPV vaccination, but the HPV inoculation had proved successful and was/is still needed [5,13]. Because HPV was strongly associated with female cervical cancer, HPV infection was deemed mainly as a sexually transmitted virus and other non-sexual activities causing infection were overlooked.

Kissing

Consequently, kissing in all its forms were identified as a prime source of viral transmission of HPV. Maternal, infant, sibling and family affections are frequently and innocently expressed with kissing. Intra-familial kissing constrains the types of viral transmission, but kissing strangers are not without dangers for contracting potentially pathogenic viruses. The 'Saviolum Kiss' in which oral fluids with an open mouth are exchanged (the French kiss or Tonsil-washer), or wet-lip to skin contact allows for easy transfer of HPV's [14,15,16]. Besides oral-oral kissing, oro-genital contact (as cunnilingual or fellatio) is also identified as a prime source of viral infection [16].

Conclusion

Effective at preventing oncogenic change, not only for genital cancers, but also effective for other head and neck period neoplasias[16]. Considering recent

research, kissing is acknowledged as an important and significant contributing factor in spreading HPV through non-genital contact.

Abbreviations

HSV =Herpes Simplex Virus

EBV=Ebstein-Barr Virus

CMV = Cytomegalo Virus

HPV = Human Papilloma Virus

LCR = HPV16 long control region (LCR) sequences, for HPV-16

Five groups: European (E), Asian (As), Asian-American (AA), African 1 (Af-1), and African 2 (Af-2).

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